Journal of Cardiovascular Disease Research

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 03, 2023

A Study of Electrocardiographic Changes in Acute Cerebrovascular Accidents

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Abstract

Background: Cardiac abnormalities occur in 60 to 70 percent of patients after stroke. The most common disturbance include ECG abnormalities, cardiac arrhythmias, and myocardial injury and dysfunction distinguishing cardiac abnormalities directly caused by stroke. More importantly, cardiac disturbances are the most common cause of death in stroke accounting for up to 6 percent of unexpected death during the first month. The severity of the neurological injury is strongly associated with the presence of left ventricular dysfunction. Similarly, diastolic dysfunction is also common after SAH, is associated with the severity of the neurological injury, and maybe the cause of pulmonary edema seen in these patients. To study the incidence and pattern of ECG changes in a patient with cerebrovascular accidents To assess the relation of ECG changes in an acute cerebrovascular accident to the location of the cerebral lesion. **Material and Methods:** This is an observational study. A total of 50 patients were included in the study. The present study was conducted in a tertiary care hospital. The study was conducted at Sri Muthukumaran Medical College Hospital And Research Institute, Chikkarayapuram, Kundrathur Road, Near Mangadu, Chennai - 600 069. All patients admitted to the medical ward with acute cerebrovascular accidents who satisfy the inclusion criteria were enrolled in the study. All patients with acute cerebrovascular accidents were studied. They were assessed with serum electrolytes, X-ray and blood urea, and sugar 12 lead ECG was taken and monitored on the day of admission. CT scan was taken within 24-48 hrs. Patients were categorized based on the CT finding as cerebral infarction, cerebral haemorrhage , and subarachnoid haemorrhage . ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude, and morphology, and the QT interval was calculated. QTC interval was calculated based on Bazetts formulae. **Results:** Stroke was most common in 5th and 6th decade. Cerebral infarction formed the largest group. Males had higher preponderance. Hypertension was the most common risk factor. In total, 74% had electrocardiographic abnormality. ECG changes are more common among cerebral hemorrhage and subarachnoid hemorrhage. Most common ECG abnormality was prolonged QTc interval. Overall immediate mortality was 23%. It was high in cerebral hemorrhage. Morality was high in patients with abnormal ECG, mostly with prolonged QTc and with T-wave inversion. **Conclusion:** Patients with cerebrovascular accidents often have abnormal ECG in the absence of known organic heart disease or electrolyte imbalance. These ECG changes are more common in hemorrhagic than ischemic stroke. The mortality in these patients did not relate to the ECG changes seen but was dependent on the type of cerebrovascular accident and the level of consciousness on admission.

Keywords: Cerebrovascular accidents, electrocardiographic changes, mortality.

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 03, 2023

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Introduction

Cardiac abnormalities were described with various CNS diseases including seizures, trauma, ischemic stroke, ICH and less commonly tumors, electroconvulsive therapy and meningitis. More recently emotion and stress induced cardiomyopathy has been described. [1,2] Electrical stimulation experiments suggests a posteriorly located area of cardiovascular sympathetic control and anterior parasympathetic control region. [3,4] Asehenbrenner and Bodeehte,[5] is reported that intracranial lesions may be responsible for electrocardiograph changes, but the first report of ECG abnormalities in patients with cerebrovascular accidents was given by Byer, Ashman and Toth. Burch, Myers, and Abildskov stated a pattern of QT prolongation, abnormal T waves, and U waves which they considered unique of acute cerebrovascular stroke. [5] The physiological & anatomical pathways involved in brain-heart interaction have been explained in both animal and human studies. ‘‘Ability to propagate the arrhythmia by activation of the sympathetic nervous system represented a neurogenic mechanism’’.[6] The medulla oblongata has been described as the principal site of vagal sympathetic and parasympathetic region involved in cardiac control.[7] In addition both anatomical and physiological evidences implicate the hypothalamus in cardiac control.[8] The prevalence of all types of stroke was 12%, among 0.2% cerebrovascular stroke was occurred in younger age group in Indian population.10 It is estimated that1.2% to 2.4% of mortality occurred in younger age to older age in the country.[9,10] ECG abnormalities occurred in 60-90% of patients with intraparenchymal or subarachnoid bleed and in about 5-20% of patients with acute ischemic stroke. The underlying basis is disordered repolarization process. [11] The possible mechanism is through disruption in autonomic regulation and massive stimulation of the sympathetic nervous system. [12] An observational study of stroke patients indicated an increased incidence of sudden death among patients with right insular. strokes. In view of the varied explanation for the ECG abnormalities in acute CVA, the present study was undertaken to review the pattern of ECG changes associated to pathophysiologic categories of acute stroke among patients without cardiovascular disease and to determine if specific ECG changes are related to the location of lesions. [13]

Review of Literature

Cerebrovascular disease includes some of the most common devastating disorders ischemic stroke, hemorrhagic stroke, and cerebrovascular anomalies such as intracranial aneurysm and AV malformation. They cause 2000000 deaths each year in developed countries such as the USA and major cause of disability. Accounts for 10% of all strokes associated with a 50% case fatality rate. Hypertension, trauma, and cerebral amyloid angiopathy cause major hemorrhage. Aneurysmal subarachnoid hemorrhage and hypertension intracranial hemorrhage are important causes. Common site are basal ganglia, (putamen), thalamus cerebellum and pons. In non-hypertensive patients, hemorrhagic disorders, neoplasm, and vascular malformation are the causes. The hemorrhage may be small or large, a clot may form and compress adjacent tissue causing herniation and death blood may dissect into ventricular space which increases morbidity and cause hydrocephalus. Ako J, Sudhir K et al,[1] stressed the absence of any clinical evidence of coronary infarction and normality of the heart and coronary arteries at autopsy. Thus, there is a marked difference in the reported frequency of ECG abnormalities in patients with CNS lesions. Cardiac arrhythmias can be detected in almost all patients during the first few hours after SAH; in approximately 20% of

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 03, 2023

cases, the arrhythmias can be severe or life-threatening. Ventricular arrhythmias are a potential cause of sudden death after SAH. By bee KA,[2] noted torsades de pontes in 3. 8% of 132 patients with SAH who underwent Holter monitoring. Increased QT dispersion is a common electro-cardiographic finding after SAH. Changes resembling those seen in acute myocardial ischemia can be noted in 25% to 80% of patients. Many people with SAH have secondary myocardial ischemia and left ventricular dysfunction. An elevation of the cardiac isoenzyme creatine kinase can be detected. Sub endocardial areas of focal ischemic necrosis are found among patients who died of SAH even those without prior history of coronary artery disease. The reduction of cardiac output after severe SAH might increase the risk of cerebral ischemia secondary to vasospasm. Wittstein IS et al,[3] stated that the relative balance of excitatory and inhibitor pathways involved in cardiovascular control can vary with the state of arousal as well as with the involvement of adjacent inhibitory pathways in the frontoparietal cortex. Evidence suggests that in humans, stroke isolated to the left anterior insula or the right frontoparietal cortex sparing the insula will have similar effects on cardiovascular outcomes. Levy A. et al,[4] These ECG changes were not associated with any particular site of the cerebral lesion. Cardiac disturbances are diverse and frequent in the setting of acute neurological injury. More importantly, the presence of cardiac abnormalities has a significant impact on clinical management and affects cardiac and neurological outcomes.

Methodology

Study Population; Patients Attending the General Medicine OPD of Sri Muthukumaran Medical College Hospital and Research Institute, Chikkarayapuram, Kundrathur Road, Near Mangadu, Chennai - 600 069.

Place of the study: Sri Muthukumaran Medical College Hospital and Research Institute, Chikkarayapuram, Kundrathur Road, Near Mangadu, Chennai - 600 069

Duration of the study: 8-10 months after ethical committee approval

Study design: Descriptive cross-sectional study,

Sample Size: Random sampling method (50 patients)

Sampling technique: Convenient sampling.

Inclusion criteria

- Age above 30 years.
- All patients with acute cerebrovascular accidents admitted to the medical ward within 72 hrs. of the onset of stroke were considered. After admission, a detailed history regarding the temporal profile of the stroke including the history of risk factors like hypertension, diabetes mellitus, smoking, history of ischemic heart disease, and rheumatic heart disease was obtained

Exclusion Criteria

- Patients showing cardiomegaly on X-ray were excluded from the study.
- Patients previously diagnosed to have electrolyte abnormalities were also excluded from the study
- Patients with a history of underlying heart diseases previously diagnosed with ECG abnormalities and hepatic or renal diseases are excluded from the study.
- Patients were categorized into 3 different cardiovascular diseases based on the CT finding cerebral infarction, cerebral hemorrhage, and subarachnoid hemorrhage.

Data collection method

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 03, 2023

Well-constructed Performa in the English language containing various aspects of information about patients which include demographic details, Personal habits, clinical presentations, laboratory investigation, and treatment will be used to collect the details from the participant (patient)

Statistical analysis

ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude, and morphology, and the QT interval was calculated. QTc interval was calculated based on Bizet's formula. The data obtained will be tabulated and subjected to statistical analysis using SPSS software. Study variables will be described and comparisons will be made Chi-square test.

RESULTS

In our study showed that the majority of cases (84%) were seen in 51-70 years of age groups [Table 1]. Out of 22, 12 (57%) of patients with hemorrhages, 9 (34%) of patients with infarct had changes and 1 (33.33%) patient with SAH had changes [Table 2]. ST segment changes were most commonly noted after cerebral hemorrhage. 31% of patients with infarction had ST depression. ST elevation was found in 33.33% of patients with ICH [Table 3]. T wave changes were present in 52.38% of patients with ICH. 27% of patient with infarct had T wave changes [Table 4]. The mean value of QT and QTC interval in infarction was 0.3719±0.04 & 0.4370±0.06 respectively and in hemorrhage was 0.4205±0.08 & 0.456±0.054 respectively [Table 5]. Rhythm disturbance were present in 11.53% of patients with infarct. 38% of patients with ICH have changes of which 14.28% had sinus tachycardia and 24% had sinus bradycardia. 33.33% of patients with SAH have ECG changes [Table 6].

Table 1: Age wise Distribution of Cases

Age group (yrs.)	Number of patients	Percentage
40-50 yrs.	6	12%
51-60 yrs.	19	38%
61-70 yrs.	23	46%
>70 yrs.	2	4%
Total	50	100%

Table 2: Incidence of abnormal ECG'S in the study group

Study group	No. of cases	Abnormal cases	Percentage
Cerebral Infarction	26	9	34.61%
Cerebral Haemorrhage	21	12	57.14%
Subarachnoid haemorrhage	3	1	33.33%
Total	50	22	44%

Table 3: The Incidence of ST Segment Changes in the Study Group

Study group	Total no. of cases	ST Segment Elevation	ST Depression Segment	Percentage with ST Segment Changes
Cerebral Infarction	26	1 (3.84%)	8 (30.76%)	9(34.61%)
Cerebral Haemorrhage	21	9 (42.85%)	3 (14.28%)	12 (57.14%)
Subarachnoid haemorrhage	3	1 (33.33%)	0 (0%)	1(33.33%)

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 03, 2023

Table 4: the incidence of t wave changes in the study group

Study group	Total no. of cases	Tall T Wave	T Wave Inversion	Percentage With T Wave Changes
Cerebral Infarction	26	2 (7.69%)	5 (19.23%)	7(26.92%)
Cerebral Haemorrhage	21	8 (42.85%)	3 (14.28%)	11 (52.38%)
Subarachnoid haemorrhage	3	1 (33.33%)	0 (0%)	1(33.33%)

Table 5: the mean value of qt & qt complex in study group

Study group	QT (Mean±SD)	QTC (Mean±SD)
Cerebral Infarction	0.3719±0.04	0.4370±0.06
Cerebral Haemorrhage	0.4205±0.08	0.456±0.054
Subarachnoid haemorrhage	0.3367±0.045	0.4567±0.1343

Table 6: incidence of rhythm disturbances in the study group

Study group	Total no. of cases	Sinus Tachycardia	Sinus Bradycardia	Percentage With T Wave Changes
Cerebral Infarction	26	3 (11.53%)	0 (0%)	3(11.53%)
Cerebral Haemorrhage	21	3 (14.28%)	5 (23.80%)	8 (38.09%)
Subarachnoid haemorrhage	3	1 (33.33%)	0 (0%)	1(33.33%)

DISCUSSION

Our study observed that mostly cases (84%) were seen in 51-70 years of age groups, because CVA patients admitted in hospital more than 40 years of age group during study period. The proportion of stroke death increases with age, 2.4% of all deaths in old age (>70 years of age).15 Our study compared with Bozluolcay M et al. (2003)16 mean age was 65.5 ± 11.9 (range yrs.91 yrs.). T wave inversion was observed in 14% of patients with intracerebral haemorrhage and 29% patients with cerebral infarction in our study. The mean value of QT and Q-Tc interval in infarction was 0.3719±0.04 & 0.4370±0.06 respectively and in haemorrhage was 0.4205±0.08 & 0.456±0.054 respectively. A study done by Ashman and Toth, in 1947.6 In 1954, Burch, Myers, and Abildskov 7 stated a pattern of QT prolongation, abnormal T waves, and U waves which they considered unique of acute cerebrovascular stroke. Mansoureh Togha et al (2013) reported ‘‘ECG abnormalities associated with stroke were T-wave abnormalities, prolonged Q-Tc interval and arrhythmia, which were respectively found in 39.9%, 32.4%, and 27.1% of the stroke patients’’. Dr. Abhilash Somasundaran et al (2015) reported ECG changes included T inversions (22.3%) and ST depressions (17.2%) predominantly. The next common abnormality noted was tall T waves, which was observed in 43% of patients with intracerebral haemorrhage. Our findings consisted with Byer and colleagues (1947) reported marked QT prolongation with large T and U waves on the ECG of four patients with stroke.6 The most common abnormality noted was ST segment changes (57%) in patient with cerebral haemorrhage. Of which 43% had ST segment elevation and 14% had ST segment depression. The findings were conflict with Dimant J, Grob D (1977)who found that CVA had a 7- to 10-fold higher incidence of ST segment depression.Kono and colleagues performed detailed cardiac evaluation of patients with acute SAH and reported that ST elevation in the ECG may be due to apical wall motion abnormalities on the echo- cardiogram, but there was no evidence of coronary artery stenosis or coronary artery vasospasm on angiography.20Rhythm disturbance were present in 11.53% of patients with infarct. 38 % of patients with ICH have changes of which 14.28% had sinus

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tachycardia and 24% had sinus bradycardia. 33.33% of patients with SAH have ECG changes. Our comparison with study done by Goldstein's observed bradycardia in 8% and tachycardia in 2% of patients with acute stroke. Stober et al described sinus bradycardia in 23%. Regarding the relationship between the locations of CVA lesions and ECG abnormalities, Frentz and Gormsen (1962) ‘‘ECG changes appeared to bear no relationship to arteriographic findings’’. [14,15]

CONCLUSION

The changes found in ECG of cardiovascular disease patients were not associated with any particular site of cerebral lesion. Cardiac disturbance are diverse and frequent in the setting of acute neurological injury. Furthermore, the presence of cardiac abnormalities has significant impact on clinical management and affects cardiac and neurological outcome. Understanding that these ECG changes which are occurring in patients with cerebrovascular accidents is important because it may lead to erroneous judgment of assigning these patients as cardiac dysfunction.

Limitations:

The present will demonstrate that by continuous ECG monitoring cardiac disturbances, especially rhythm and conduction abnormalities can be detected and treated immediately. We didn’t induce ECHO cardiography.

REFERENCES

1. AkoJ, SudhirK, Farougue HM, et al. Transient left ventricular dysfunction under severe stress: brain-heart relationship revised. Am J Med. 2006; 6(2):119-10.
2. By bee KA, Kara T, Prasad A, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. Ann Intern Med. 2004; 14(1):858-859.
3. Wittstein IS, Thiemann DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005 35(2):539.
4. Levy A. The exciting causes of ventricular fibrillation in animals under chloroform anaesthesia. Heart 1913;4(1):319.
5. Levy A. Further remarks on ventricular extra systoles and fibrillation under chloroform. Heart 1919;7(2):105,53.
6. Lane RD, Wallace JD, Petrosky PP, et al: Supra ventricular tachycardia in patients with right hemisphere strokes. Stroke 1992;23(4):362.
7. Vingerhoets F, Bogousslavsky J, Regli F, VanMelle G. Atrial fibrillation after acute stroke. Stroke. 1993;24(2):26.
8. Algra A, Gates PC, Fox AJ, et al. Side of brain infarction and long-term risk of sudden death in patients with symptomatic carotid disease. Stroke 2003;34(5):2871.
9. Oppenheimer S: cerebrogenic cardiac arrhythmias: cortical lateralization and clinical significance. Clin Auton Res 2006;16(6):6.
10. Kono T, Morita H, Kuroiwa T, et al. Left ventricular wall motion abnormalities in patients with subarachnoid haemorrhage: neurogenic stunned myocardium. JAm Coll Cardiol 1994;24(1):636.
11. Kuroiwa T, Morita H, Tanabe H, et al. Significance of ST segment elevation in electrocardiograms in patients with ruptured cerebral aneurysms. Act a Neurochir; 1995:14(2)133-141.
12. Lindgren A, Wohlfart B, Pahlm O, Johansson BB. A Study of ECG changes in stroke patients without primary heart disease. Department of Neurology, University Hospital, Sweden.1994.

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13. Arrudoand Lacerda. ECG findings in acute cerebrovascular haemorrhage neuropsychiatry 1992;50(3):269-74.
14. Keller and Williams. Cardiac dysrhythmias associated with CNS dysfunction; Journal of Neuroscience 1993;25(3)49-55.
15. Cruickshnok, Neil Dcoyerand J. Price. ECG changes and their prognostic significance in CVA–Journal of Neurology 1974;37(5):735-759.